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10/554,123	08/21/2006	Marja T. Nevalainen	G0762.70004US01	4634
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WOLLENBERGER, LOUIS V				
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary

Application No.

10/554,123

Applicant(s)

NEVALAINEN, MARJA T.

Examiner

Louis Wollenberger

Art Unit

1635

Period for Reply -- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 29 December 2008.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1, 4-7, 11, 13, 15-21, 25-28, 30-33, 37, 39 and 41-53 is/are pending in the application.
- 4a) Of the above claim(s) 4-7, 15-20, 25, 26, 30-33, 41-46 and 48-52 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1, 11, 13, 21, 27, 28, 37, 39, 47 and 53 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☐ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-846)
- 3) ☒ Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date 12/29/2008
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date _____
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: _____

DETAILED ACTION

Status of Application/Amendment/Claims

Applicant's response filed 12/29/2008 has been considered. Rejections and/or objections not reiterated from the previous office action mailed 8/28/2008 are hereby withdrawn. The following rejections and/or objections are either newly applied or are reiterated and are the only rejections and/or objections presently applied to the instant application. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Applicant's amendment to the claims, filed 12/29/2008, are acknowledged. With entry of the amendment, claims 1, 4-7, 11, 13, 15-21, 25-28, 30-33, 37, 39, and 41-53 are pending. Claims 4-7, 15-20, 25, 26, 30-33, 41-46, and 48-52 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim.

Claims 1, 11, 13, 21, 27, 28, 37, 39, 47, and 53 are examined herein.

Specification

Applicant's submission of the statement that the substitute specification filed 10/21/2005 includes no new matter, as required under 37 CFR 1.125, is acknowledged. The substitute specification filed 10/21/2005 (49 pages) has been entered into the application.

Claim Objections

Claim 37 is objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the

claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form.

The claims recites the method of claim 27, wherein the siRNA inhibits the expression of a Stat5b polypeptide. The use of the indefinite article "a" indicates the claim is drawn to the inhibition of any Stat5b polypeptide when only one Stat5b polypeptide is clearly embraced by the method of claim 27. Further, neither the prior art nor the specification teach that multiple Stat5b polypeptides exist. Replacing "a" with "the" would be remedial.

Claim Rejections - 35 USC § 112, first paragraph (enablement)

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1, 11, 13, 21, 27, 28, 37, 39, 47, and 53 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for

a method of inhibiting the growth of prostate cancer cells in which endogenous Stat5 is active, and for a method of treating prostate cancer in a male, wherein said cancer comprises cells in which Stat5 is active, by inhibiting the activity or expression of Stat5 (i.e., both Stat5a and Stat5b) using an siRNA that inhibits Stat5a and Stat5b, does not reasonably provide enablement for

a method of inhibiting the growth of prostate cancer cells in which endogenous Stat5 is inactive, or for a method of treating prostate cancer in a male, wherein the cancer does

not comprise cells that are Stat5 active, by inhibiting the activity or expression of any Stat5 (a or b), or for any method for inhibiting the growth of any prostate cancer cell or treating prostate cancer due to any cell type by inhibiting the activity of Stat5b only.

The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

Factors to be considered in a determination of lack of enablement include, but are not limited to:

- (A) The breadth of the claims;
- (B) The nature of the invention;
- (C) The state of the prior art;
- (D) The level of one of ordinary skill;
- (E) The level of predictability in the art;
- (F) The amount of direction provided by the inventor;
- (G) The existence of working examples; and
- (H) The quantity of experimentation needed to make or use the invention based on the content of the disclosure.

In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)

The invention involves gene therapy and is in a class of invention that the CAFC has characterized as “the unpredictable arts such as chemistry and biology.” *Mycogen Plant Sci., Inc. v Monsanto Co.*, 243 F.3d 1316, 1330 (Fed. Cir. 2001).

Claims 1 and 27 are representative of the methods now claimed. The preambles of the methods state intended uses and describe specific effects to be achieved by performing the steps recited in the body of the claims. The language therefore requires the instant claims be evaluated to determine whether the instant application enables one of skill to practice the instant methods in the manner intended to achieve these effects without having to engage in undue experimentation.

US Provisional Application 60/465014, to which priority is claimed, teaches Stat5 exists as two highly homologous isoforms, Stat5a and 5b, which have more than 95% amino acid homology and are encoded by separate genes (page 7). The disclosure further teaches that dominant-negative Stat5 gene therapy induced cell killing in cells where endogenous Stat5 was active (e.g. CWR22, LNCaP) but not in cells where endogenous Stat5 was not active (e.g. PC-3). Beginning at page 13, the prior filed application shows how to inhibit the growth (and induce apoptosis) of prostate cancer cells in which endogenous Stat5 is active, by inhibiting both Stat5a and Stat5b simultaneously using a recombinant expression construct, DNStat5, which is said to suppress both Stat5a and Stat5b-mediated transcriptional activation. Beginning at page 16, the prior filed application shows how to detect Stat5 activation in paraffin embedded tissue (as might be obtained by biopsy). It is shown that Stat5a and Stat5b are activated in some prostate cells but not all prostate cells. It is shown that expression of DNStat5 induces cell death in cells in which Stat5a and b are active but not in cells in which Stat5a and b are not active.

Little or no direction is found in the prior art relating to the inhibition of Stat5 polypeptides for the treatment of prostate cancer in males or even to the inhibition of prostate

cancer cell growth in culture or in animals. Moreover, there is no evidence in the prior art teaching or suggesting that inhibition of Stat5b alone will provide for any of the effects now claimed.

For example, Ahonen et al. (2002) *Endocrinology* 143(1):228-230 teach that PRL signaling in rat prostate tissue, which may be involved in the development of prostate tumors, is primarily transduced via Stat5a and Stat5b. The reference further teaches Stat5a and Stat5b are expressed to varying degree in separate lobes of rat prostate. However, Ahonen et al. do not teach that inhibiting one Stat5 isoform or the other alone will inhibit prostate cancer cell growth or have therapeutic benefit to any male suffering from prostate cancer. Rather, this reference like the instant application teaches that Stat5a and b are both involved in prostate cell viability. Indeed the instant application teaches that inhibition of Stat5 activity in general, as by the suppression of both known isoforms, a and b, causes prostate cancer cell death in cells in which Stat5 is active. Neither the prior filed application nor the instant applicant shows how to inhibit the growth (and/or induce apoptosis) of prostate cancer cells in which endogenous Stat5 is inactive. Further, neither the prior filed application nor the instant application teaches one of skill how to inhibit the growth (or induce apoptosis) of any prostate cancer cell (i.e., stat5 active and inactive cells) or how to treat prostate cancer due to Stat5 active or inactive cells in any individual by inhibiting Stat5b only.

While the inhibition of Stat5-active prostate cancer cell growth in vitro by expression of a dominant-negative protein that inhibits the activity of both isoforms of Stat5 may be correlative of a method of treating Stat5-active prostate cancer in a male by inhibiting the expression of both

Stat5a and Stat5b using an siRNA, and while the instant claims may embrace such methods, the instant methods also explicitly recite methods for accomplishing these effects by inhibiting Stat5b only in both Stat5-active and -inactive cells, which is not adequately represented by the prior filed application or the prior art. There is no evidence of record to show or even suggest that inhibiting the Stat5b isoform only will produce the effects now claimed. Therefore, it is reasonable to question the objective truth of the statements in the claims.

It has been pointed out by the USPTO Board of Patent Appeals and Interferences in *Ex parte De La Monte et al.* (2006) (Appeal No. 2006-0275; Application No. 09/964,667) that

"... as explained in PPG Indus., Inc. v. Guardian Indus. Corp., 75 F.3d 1558, 1564, 37 USPQ2d 1618, 1623 (Fed. Cir. 1996), undue experimentation has little to do with the quantity of experimentation; it is much more a function of the amount of guidance or direction provided..."

In the instant case, the examples and general statements in the instant disclosure do not provide the direction or guidance necessary to enable one of skill in the art to achieve the effects required by the claims without resorting to trial and error experimentation, with no assurance of ever reaching a successful conclusion.

Therefore, considering the breadth of the claims, the state of the art at the time of filing, the level of unpredictability in the art, and the limited guidance and working examples provided by the instant application, the Examiner submits that the skilled artisan would be required to conduct undue, trial and error experimentation to practice the claimed invention commensurate with the claims scope. Accordingly, the instant claims are rejected for failing to comply with the enablement requirement.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

The factual inquiries set forth in *Graham v. John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1, 11, 13, and 21 are rejected under 35 U.S.C. 103(a) as being unpatentable over Shaw et al. (WO 03/026641) in view of:

1. Leong et al. (2002) *Oncogene* 21:2846-2853;
2. Turkson et al. (2000) *Oncogene* 19:6613-6626;
3. Ahonen et al. (2002) *Endocrinology* 143:228-238; and
4. Tuschl et al. (US 20040259247 A1).

Claim interpretation:

MPEP 2111.02 states if the body of a claim fully and intrinsically sets forth all of the limitations of the claimed invention, and the preamble merely states, for example, the purpose or intended use of the invention, rather than any distinct definition of any of the claimed invention's limitations, then the preamble is not considered a limitation and is of no significance to claim construction. *Pitney Bowes, Inc. v. Hewlett-Packard Co.*, 182 F.3d 1298, 1305, 51 USPQ2d 1161, 1165 (Fed. Cir. 1999). During examination, statements in the preamble reciting the purpose or intended use of the claimed invention must be evaluated to determine whether the recited purpose or intended use results in a structural difference (or, in the case of process claims, manipulative difference) between the claimed invention and the prior art.

In the instant case the Examiner submits the preamble of claim 1 ("A method of inhibiting prostate cancer cell growth") recites an intended use only, and does not add any manipulative or structural difference to the body of the claim, which fully and intrinsically sets forth all of the limitations of the claimed invention. Accordingly, for purposes of the application of prior art, the preamble in claim 1 is given no patentable weight.

As now written, claim 1 requires nothing more than inhibiting Stat5b activity in a prostate cancer cell by contacting said cell(s) with an siRNA that inhibits Stat5b activity.

The effect recited in the preamble is considered to be a property inherent to the method step of contacting prostate cancer cells with an siRNA targeted to Stat5b.

The express, implicit, and inherent disclosures of a prior art reference may be relied upon in the rejection of claims under **35 U.S.C. 102** or **103**. "The inherent teaching of a prior art

reference, a question of fact, arises both in the context of anticipation and obviousness." *In re Napier*, 55 F.3d 610, 613, 34 USPQ2d 1782, 1784 (Fed. Cir. 1995).

As explained below, the instantly applied references reasonably suggest targeting Stat5a and Stat5b genes in prostate cancer cells in vitro and in vivo for research purposes to investigate the role these polypeptides play in prostate cancer cell growth. While the prior art may not disclose that inhibiting one or both of these polypeptides will result in prostate cancer cell death or inhibit prostate cancer cell growth, the combination of references as whole reasonably suggest the method recited in the body of the claims for inhibiting Stat5b to further understand the function of Stat5b in prostate cancer cells, as it is the normal desire of the scientist to understand the function of every protein, particularly those that may be involved in cell transformation; therefore one of skill performing the suggested method would necessarily obtain the effects inherent to the method, including those recited in the claims, as evidenced by the claims. Burden is shifted to Applicant to show otherwise (MPEP 2112).

The rejection:

Shaw et al. disclosed and claimed a methods for making and using Stat5a and Stat5b inhibitors to treat prostate carcinoma (page 3, 4, 6, 7, and claim 4, page 44). [Note: Shaw et al. use the term "prostrate carcinoma", which is believed to be an obvious typographical error for the intended term "prostate carcinoma."]. In addition, Shaw et al. specifically recommend and teach using an inhibitor of Stat5b to treat prostate carcinoma. Accordingly, Shaw et al. clearly suggested a link between Stat5 polypeptide expression and/or activity and prostate carcinoma,

and therefore provide reason to study Stat5 polypeptide activities as they may relate to prostate cancer cell transformation and growth.

In addition to Shaw et al., several reports in the prior art literature taught and suggested the involvement of Stat5 in a number of different cancer, and that the inhibition of Stat5 proteins may, in some cases, inhibit cancer cell growth.

For example, Leong et al. taught that targeting Stat5b using antisense oligonucleotides inhibits squamous cell carcinoma of the head and neck (pp. 2846-2853).

Turkson et al. taught that STAT family members, including Stat3 and Stat5, are associated with a wide variety of human malignancies, including breast, head and neck, and prostate cancers (pp. 6613-6626, Table 1).

Ahonen et al. taught that PRL signaling in rat prostate tissue, which may be involved in the development of prostate tumors, is primarily transduced via Stat5a and Stat5b, and that therapy-based killing of prostate cancer cells may require combined blockade of distinct signaling pathways of several growth factors and cytokines, among which Stat5 proteins may provide a good candidate target.

Accordingly, the prior art is replete with disclosure suggesting a link between Stat5b polypeptide activity and cancer, including prostate cancer. As a result, it would have been the normal desire of one of skill in the art at the time of invention to make and use Stat5b inhibitors to inhibit Stat5b expression in any cancer cell, including prostate cancer cells, in vitro or in vivo, to further investigate the role of Stat5b in cancer cell growth in vitro and in vivo. Given the ample disclosure suggesting Stat5b may be involved in many types of cancers, the practitioner would have had reason to study Stat5b activity in virtually any cell type prone to cancer, in

which Stat5b is actively expressed, including any prostate cancer cell line known in the art at the time, such as the human prostatic carcinoma cell line LNCaP.

While Shaw et al. does not teach siRNA for inhibiting Stat5b activity, it would have been obvious to one of skill in the art at the time, interested in investigating Stat5 polypeptide function in prostate carcinoma, to inhibit Stat5a and b polypeptide activities using any method known in the art for inhibiting protein activity, including any of the established loss-of-function techniques readily available at the time, including RNA interference.

The level of skill and knowledge in the art of RNA interference in mammalian cells at the time of invention was such that the design, preparation, and use of short interfering RNAs (siRNA) against a known gene was conventional, as evidenced by Tuschl et al., who provide a complete blueprint for the design, synthesis, and application of short interfering RNAs for sequence-specific inhibition of gene expression in a mammalian cell in vitro and in vivo, and expressly recommend using siRNAs to investigate gene/protein function in healthy and diseased cells and tissues.

In view of the wealth of guidance and direction suggesting and teaching that Stat5 proteins such as Stat5b cause or promote cancer cell growth, or, at the least, represent potential therapeutic targets, given that in at least one case in the prior art showed that inhibition of Stat5b inhibits cancer cell growth (Leong et al.), and given that the prior expressly taught and suggested inhibiting Stat5b to treat prostate carcinoma (Shaw et al. and Ahonen et al., respectively), one of skill would have had ample reason to make and use siRNAs targeted to Stat5b to inhibit Stat5b in any prostate cancer cell line to learn more about the role Stat5b polypeptides play in prostate cell transformation and cancer cell growth, as well as to determine the therapeutic potential of

methods that inhibit Stat5b, with regard to cancers, including prostate cancer. Given the direction and guidance in the prior art relating to the use of siRNA to inhibit mammalian genes in mammalian cells in vitro and in vivo, as shown by Tuschl et al., one of skill would have expected to successfully inhibit Stat5b in any cell in which it was actively expressed. As a result of such inhibition in prostate cancer cells expressing Stat5b, one of skill would have necessarily obtained an inhibition of prostate cancer cell growth, since such effects are, as evidenced by the claims, inherent to such methods.

Accordingly, in the absent of convincing evidence to the contrary, the instantly claimed invention would have been *prima facie* obvious to one of skill in the art at the time the invention was made.

Response to Arguments

The portion of Applicant's reply filed 12/29/2008 deemed pertinent to claims 1, 11, 13, and 21 is addressed herein.

Applicant argues the combination of references do not suggest or teach methods of treating prostate cancer using Stat5b inhibitors. The Examiner agrees only to the extent the instant combination of references (considered to be the closest prior art) does not reasonably enable one of skill to treat prostate cancer with reasonable success. However, the prior art does suggest a link between Stat5b and cancer, including prostate cancer, and this is considered sufficient to provide reason for one of skill to study Stat5b activity in any cancer cell, including prostate cancer cells, using any research tool available at the time, including RNA interference. Furthermore, claims 1, 11, 13, and 21 do not require treatment of prostate cancer or even *a priori* knowledge of whether Stat5b activity promotes or even influences cancer cell growth, and the

claims read on method of inhibiting Stat5b in prostate cancer cells using siRNA for any reason, such as for basic research to establish the role of Stat5b activity in prostate cancer cells.

Similarly, Applicant argues the inadequacy of the Ahonen, Leong, and Turkson references on many fronts, generally arguing the references fail to establish that inhibition of Stat5b will induce prostate cancer cell death, and asserting passages cited in the Office Action are mischaracterizations or speculative statements. While many of Applicants arguments are persuasive as they relate to claim 21 and claims dependent thereon, drawn to a method of treating, the arguments are not persuasive as they relate to claims 1, 11, 13, and 21, which require nothing more than a reason to inhibit Stat5b in prostate cancer cells and the direction needed to make and use siRNA inhibitors of Stat5. The applied references as a whole clearly suggested the link between Stat5b and many types of cancer, including, possibly, prostate. It would not have been necessary for one of skill at the time to have known that inhibiting Stat5b would inhibit prostate cancer cell growth; one of skill would have had reason to make and use siRNAs against any known gene simply for the purpose of establishing the function of that gene in any cell. One would reasonably have targeted Stat5b in prostate cancer cells given the guidance in the literature suggesting Stat5b may be involved in cancer in many cells, including prostate.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Louis Wollenberger whose telephone number is (571)272-8144. The examiner can normally be reached on M-F, 8 am to 4:30 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James (Doug) Schultz can be reached on (571)272-0763. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Louis Wollenberger/
Examiner, Art Unit 1635
February 23, 2009